# Height as a Marker of Childhood Development and Late-life Cognitive Function: The Honolulu–Asia Aging Study

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**ABSTRACT.** *Objective.* **Growing evidence suggests that structural and functional brain reserves, thought to develop in childhood and adolescence, may be crucial in determining when cognitive impairment begins. The purpose of this report is to examine the relationship of height, as a marker of childhood development, to late-life cognitive function in a sample of elderly Japanese-American men.**

*Method.* **Cognitive performance was assessed from 1991 to 1993 in the Honolulu–Asia Aging Study in 3733 men aged 71 to 93 years and related to height that was measured 25 years earlier.**

*Results.* **Among the study sample, shorter men were older, leaner, and less educated than taller men. Shorter men also spent more years of their childhood living in Japan and were more likely to have had fathers in unskilled professions. After adjustment for age, the prevalence of poor cognitive performance declined consistently with increasing height from 25% in men shorter than 154 cm (61 in) to 9% in those taller than 174 cm (69 in). Excluding men with stroke or dementia did not alter the association between height and cognitive performance. Apolipoprotein E4 was unrelated to height and did not effect the association between height and cognitive function. The prevalence of Alzheimer's disease was higher in men who were 154 cm (61 in) or shorter as compared with men who were taller (4.7% vs 2.9%, respectively). There was no association between height and vascular dementia.**

*Conclusion.* **Efforts to improve prenatal and early life conditions to maximize growth in childhood and adolescence could diminish or delay the expression of cognitive impairments that occur later in life. Prevention of some late-life cognitive impairments may have pediatric origins.** *Pediatrics* **1998;102:602–609;** ***dementia, cognitive function, childhood development.***

Although the processes leading to dementia are complex, specific factors that modulate its clinical expression are almost entirely undefined. There is growing evidence, however, that structural and functional brain reserves, thought to develop in childhood and adolescence, may be crucial in determining when cognitive impairment begins.[1–5] If correct, these findings suggest that cognitive impairment and dementia in late life could be more frequent among elderly persons who experienced nutritional, educational, or social deprivation during childhood. Such reports further suggest that effective strategies for reducing the frequency of clinically apparent Alzheimer's disease in late life may involve maximizing brain reserves through public health measures that optimize childhood growth and development. The purpose of this report is to examine height in middle life, as a marker of childhood conditions that promote growth, in relation to late-life cognitive function and dementia in a large group of elderly Japanese-American men living in Hawaii.

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## MATERIALS AND METHODS

### Study Sample

The Honolulu–Asia Aging Study began in 1991 as a prospective study of aging and cognition in a sample of Japanese-American men who were originally enrolled in the Honolulu Heart Program from 1965 to 1968.[6] Men who comprised the original cohort were either immigrants or the progeny of immigrants from Japan who migrated to Hawaii as contract laborers in the sugar and pineapple industries. Most came from the rural districts of a few prefectures in southern Japan. Many of the men of the migrating generation remained in Hawaii at the close of their contracts where they worked as artisans, merchants, and independent farmers. The second generation continued in these trades, although others gained in affluence and worked in professions that were less physically demanding.

The original cohort included 8006 men. During the time of study enrollment (1965 to 1968), study participants received a baseline physical examination, including the collection of data on standing height, body mass index (kg/m$^2$), educational attainment, place of birth, years lived in Japan, and father's occupation. Height was measured to the nearest inch in all but 3 men. Procedures followed were in accordance with institutional guidelines and approved by an institutional review committee. Informed consent was obtained from the study participants.

Among the members of the original cohort, 3734 received screening examinations for dementia and cognitive performance 25 years later from 1991 to 1993. To account for genetic susceptibility to Alzheimer's disease,[7] frequency of apolipoprotein E4 was also determined based on plasma protein phenotyping by the Northwest Lipid Research Laboratories in Seattle, Washington. After excluding 1 man whose height was not available, analysis was based on 3733 men. Those screened for dementia comprised ~80% of the surviving members of the original cohort enrolled into the Honolulu Heart Program.

### Measurement of Cognitive Performance

Cognitive performance was assessed using the Cognitive Abilities Screening Instrument, which was developed and validated

for use in cross-cultural studies as a brief but comprehensive measure of intellectual function. It is a composite of the Folstein Mini-Mental State Examination,[8] the Modified Mini-Mental State Examination,[9] the Hasegawa Dementia Screening Scale,[10] and additional items to assess judgment and language fluency.[11,12] Scores range from 0 to 100 with 100 indicating optimal cognitive performance. For this report, poor cognitive performance is defined as a score less than 74. Scores below this value were selected a priori as an indicator of possible dementia during the first stages of dementia case-finding.[13] For men who were ultimately diagnosed as having dementia, 96% had screening scores under 74. For men without dementia, 11% had screening scores under 74. The value of 74 corresponds closely to a score of 22 on the Mini-Mental State Exam.

### Diagnosis of Dementia

Cases of dementia were identified using a three-phase screening and evaluation system that has been described previously.[13] A diagnosis of dementia was based on an evaluation by a neurologist that consisted of a history from a family informant, a standardized neuropsychologic evaluation, and a neurologic examination. Laboratory findings and computed tomography were also used for the classification of dementia. Final diagnoses were assigned by a consensus panel consisting of a neurologist and three other physicians with expertise in dementia. Participants with dementia met the criteria of the *Diagnostic and Statistical Manual of Mental Disorders* (3rd ed, revised).[14] Research criteria established by the National Institute of Neurological and Communicative Disorders and Stroke and the Alzheimer's Disease and Related Disorders Association were used in the diagnosis of Alzheimer's disease.[15] For this report, Alzheimer's disease was defined to include all persons whose diagnosis was probable or possible and was considered to be the primary cause of the dementia.[13] Diagnoses of vascular dementia used the California criteria.[16] Vascular dementia included probable or possible cases in which a vascular cause was considered the primary contributing factor.

### Statistical Methods

The relationship of height to poor cognitive performance and dementia was examined through the use of logistic regression models.[17] For comparison of cognitive performance and each dementia outcome across height strata, rates were adjusted for age and early childhood exposures including years of childhood lived in Japan, level of education, and father's occupation (skilled versus unskilled).[18] Prevalence of poor cognitive performance by height is also given for men without stroke or dementia. Here, stroke is defined to include events with either a hemorrhagic of thromboembolic origin that preceded the cognitive screening examination. Further description of the definition of stroke is provided elsewhere.[19] Proportional hazards regression models were also used to examine the effect of height on survival from the time of study enrollment (1965–1968) among all cohort members.[20]

## RESULTS

At the time height was measured (1965–1968) in the 3733 men who were later screened for dementia (1991–1993), the average age was 52.7 ± 4.7 years. The average age at the time of screening was 77.8 ± 4.7 years. In this group, 586 (16%) had poor cognitive performance and 226 (6%) had dementia. Alzheimer's disease was the sole or primary cause in 118 cases and vascular dementia was the sole or primary cause in 80 cases.

In Table 1, the distribution of age and childhood exposures are given according to height based on information that was collected at the time of study enrollment (1965–1968). In each instance, age, the percentage of men born in Japan, and the number of childhood years spent in Japan declined significantly with increasing height ($P < .001$). As compared with men who were 173 cm (68 in) or taller, those who were 152 cm (60 in) or shorter were nearly 5 years older. Nearly 20% of the shortest men were born in Japan versus only 2% of the tallest men. On average, the tallest group of men spent 6 months of their childhood in Japan as compared with more than 5 years for the shortest men.

Height was also consistently and significantly ($P < .001$) related to the percent of men whose fathers were in skilled professions and to level of education. For men who were 173 cm (68 in) or taller, 43% had fathers in skilled professions versus 23% for men who were 152 cm (60 in) or shorter. Men in the shortest group, on average, received no more than an intermediate or junior high school level of education as compared with the tallest group of men who tended to be at least high school graduates. Height was unrelated to frequency of apolipoprotein E4.

Table 2 compares the sample of men who had poor and acceptable cognitive performance based on the screening examination which occurred from 1991 to 1993. Comparisons are also made between men who were screened and not screened for dementia. Among the latter group, 3374 died before the cognitive screening examination took place. The remaining 896 men either declined to be examined or could

**TABLE 1.** Mean Age and Childhood Exposures According to Standing Height of the Men Screened for Dementia*

| Height (cm) | *n* | Age† (y) | Born in Japan (%)† | Years of Childhood Spent in Japan† | Fathers in a Skilled Profession (%)‡ | Education§ | Presence of Apolipoprotein E4 (%) |
|---|---|---|---|---|---|---|---|
| ≤152 (≤60)¶ | 163 | 55.8 | 19.0 | 5.3 | 23.0 | 2.0 | 16.2 |
| 155 (61) | 219 | 54.6 | 15.1 | 4.5 | 27.6 | 2.3 | 10.7 |
| 157 (62) | 401 | 53.7 | 10.7 | 2.8 | 30.7 | 2.5 | 15.2 |
| 160 (63) | 579 | 53.1 | 7.9 | 2.2 | 35.6 | 2.6 | 15.9 |
| 163 (64) | 677 | 52.7 | 6.2 | 2.0 | 32.6 | 2.6 | 17.7 |
| 165 (65) | 592 | 52.0 | 4.2 | 1.4 | 39.6 | 2.8 | 15.9 |
| 168 (66) | 511 | 51.9 | 4.5 | 1.2 | 40.4 | 2.8 | 16.8 |
| 170 (67) | 303 | 51.8 | 3.3 | 0.7 | 43.8 | 3.0 | 15.7 |
| ≥173 (≥68) | 288 | 51.0 | 2.1 | 0.5 | 42.9 | 3.1 | 20.2 |
| Overall | 3733 | 52.7 | 6.9 | 2.0 | 36.0 | 2.7 | 16.3 |

* Height, age, and the childhood exposures were recorded at the time of study enrollment (1965–1968). Apolipoprotein E4 was determined at the time of screening for cognitive impairment (1991–1993).
† Age, percent of men born in Japan, and years of childhood spent in Japan decline significantly with increasing height ($P < .001$).
‡ Percentage of men with fathers in a skilled profession increases significantly with increasing height ($P < .001$).
§ Education is recorded as the highest level of education achieved (0 = none, 1 = primary, 2 = intermediate or junior high school, 3 = high school, 4 = technical school, and 5 = university). Level of education increases significantly with increasing height ($P < .001$).
¶ Corresponding value in inches.

**TABLE 2.** Means and Percents of Physical Attributes and Childhood Exposures in the Men Who Were Screened for Dementia by Their Level of Cognitive Performance as Compared With the Men Who Were Not Screened But Who Were Original Cohort Members

| Attribute or Exposure‡ | Not Screened | | Screened | |
|---|---|---|---|---|
| | Status at Time of Screening | | Cognitive Performance at Screening | |
| | Alive ($n$ = 896)§ | Dead ($n$ = 3374) | Acceptable* ($n$ = 3147) | Poor† ($n$ = 586) |
| Age (y) | 53.1# | 56.7 | 52.0# | 56.5 |
| Standing height (cm) | 163# (64.1)‖ | 162# (63.9) | 164# (64.5) | 161 (63.4) |
| Body mass index (kg/m$^2$) | 23.9 | 23.8 | 23.9 | 23.7 |
| Born in Japan (%) | 7.8# | 18.1 | 4.3# | 21.0 |
| Years of childhood spent in Japan | 2.1# | 3.3# | 1.5# | 4.7 |
| Fathers in a skilled profession (%) | 36.3# | 33.4# | 37.9# | 25.7 |
| Education¶ | 2.4# | 2.4# | 2.8# | 1.9 |
| Presence of apolipoprotein E4 (%) | — | — | 16.1 | 17.8 |

* A score of 74 or higher on the Cognitive Abilities Screening Instrument.
† A score of under 74 on the Cognitive Abilities Screening Instrument.
‡ Except for apolipoprotein E4, attributes and exposures correspond to levels that were observed at the time of study enrollment (1965–1968). Apolipoprotein E4 was only determined in men who underwent screening for cognitive impairment (1991–1993).
§ Number of men.
‖ Corresponding value in inches.
¶ Education is recorded as the highest level of education achieved (0 = none, 1 = primary, 2 = intermediate or junior high school, 3 = high school, 4 = technical school, and 5 = university).
# Significantly different from men who had poor cognitive performance ($P < .001$).

not be scheduled to participate in a screening examination.

Among the men screened for dementia, those with poor cognitive performance were 4 to 5 years older and had heights that were 3 cm (1 in) less than men with acceptable cognitive performance ($P < .001$). Twenty-one percent of the men with poor cognitive performance were born in Japan as compared with 4% of men with acceptable cognitive performance ($P < .001$). Men with poor cognitive performance spent significantly more years of their childhood in Japan and were less educated ($P < .001$). Among the men with acceptable cognitive performance, 38% had fathers in skilled professions as compared with 26% of the men with poor cognitive performance ($P < .001$).

Similar comparisons exist between men with poor cognitive performance and those who were alive but not screened for dementia. Except for age and the percent of men born in Japan, this is also the case when comparing men who died before being screened for dementia with those who had poor cognitive performance. Frequency of apolipoprotein E4 was only slightly higher in men with poor versus acceptable cognitive performance.

Factors considered in Table 2 are described in Table 3 for men who were born from 1900 to 1909 versus men who were born from 1910 to 1919. For men who were screened for dementia, those born later were, on average, 3 cm (1 in) taller than men born earlier ($P < .001$). In addition to being shorter, the men who were older were also significantly leaner, although the difference was modest ($P < .001$). It was also uncommon for men in the younger

**TABLE 3.** Means and Percents of Physical Attributes and Childhood Exposures in the Men Who Were Screened for Dementia According to When the Men Were Born as Compared With the Men Who Were Not Screened But Who Were Original Cohort Members

| Attribute or Exposure* | Not Screened | | Screened | |
|---|---|---|---|---|
| | Year of Birth | | Year of Birth | |
| | 1900–1909 ($n$ = 1861)† | 1910–1919 ($n$ = 2409) | 1900–1909 ($n$ = 710) | 1910–1919 ($n$ = 3023) |
| Age (y) | 61.7‖ | 51.5¶ | 60.5# | 50.9 |
| Standing height (cm) | 161 (63.3)‡ | 164 (64.4) | 161# (63.3) | 164 (64.5) |
| Body mass index (kg/m$^2$) | 23.4 | 24.1 | 23.4# | 24.0 |
| Born in Japan (%) | 34.4 | 1.7 | 30.4# | 1.4 |
| Years of childhood spent in Japan | 5.5 | 1.2 | 5.4# | 1.2 |
| Fathers in a skilled profession (%) | 30.0 | 37.1 | 30.2# | 37.4 |
| Education§ | 2.2‖ | 2.6¶ | 2.4# | 2.7 |
| Presence of apolipoprotein E4 (%) | — | — | 14.4 | 16.8 |

* Except for apolipoprotein E4, attributes and exposures correspond to levels that were observed at the time of study enrollment (1965–1968). Apolipoprotein E4 was only determined in men who underwent screening for cognitive impairment (1991–1993).
† Number of men.
‡ Corresponding value in inches.
§ Education is recorded as the highest level of education achieved (0 = none, 1 = primary, 2 = intermediate or junior high school, 3 = high school, 4 = technical school, and 5 = university).
‖ Significantly different from men who were screened and who were born from 1900–1909 ($P < .001$).
¶ Significantly different from men who were screened and who were born from 1910–1919 ($P < .001$).
# Significantly different from men who were screened and who were born from 1910–1919 ($P < .001$).

cohort to have been born in Japan as compared with men in the older cohort (1% vs 30%; $P < .001$). Older men lived in Japan as children more than four times longer than younger men ($P < .001$). Older men were less likely to have had fathers in a skilled profession and they were less educated as well ($P < .001$). Frequency of apolipoprotein E4 was unrelated to when the men were born.

In comparisons between men within the same 10-year birth cohort (1900–1909 and 1910–1919), men who were not screened for dementia were significantly older and less educated than men who were screened ($P < .001$). Differences in the other attributes and exposures were not statistically significant.

Figure 1 illustrates the relationship between height and poor cognitive performance. After adjustment for age, the prevalence of poor cognitive performance declined consistently with increasing height ($P < .001$) from 25% in men shorter than 154 cm (61 in) to 9% in those taller than 174 cm (69 in). After removing men diagnosed with dementia and stroke, the association between height and poor cognitive performance remained ($P < .001$). Here, poor cognitive performance occurred in 18% of the shortest men and declined to 5% in those who were the tallest.

Figure 2 provides similar data for dementia (all causes combined) and for Alzheimer's disease and vascular dementia. After adjustment for age, the prevalence of dementia declined from 8% in men shorter than 154 cm (61 in) to <3% in those taller than 174 cm (69 in). Perhaps because of small numbers, the association with dementia from all causes combined is not statistically significant ($P = .101$), as is the case for Alzheimer's disease ($P = .064$) and vascular dementia ($P = .655$). Alzheimer's disease, however, was significantly more common in men 154 cm (61 in) or shorter as compared with those who were taller (4.7% vs 2.9%, respectively; $P = .018$).

To address the possibility that observations might have been the result of taller men failing to live as long as those who were shorter, data were further analyzed to describe the relationship of height to death among the entire group of men who received the baseline examination from 1965 to 1968. After adjustment for age, the association between height and mortality was absent, with men of all heights having similar risks of death.

## DISCUSSION

Growth of the brain is greatest during late gestation and in the early years of life.[21–23] Although genetics has an influence on its development, environmental factors such as malnutrition, poverty, neglect, infection, and stress may also have a role in the full expression of structural and functional brain reserves.[24–26] Maternal height, as a genetic or environmental marker, has also been related to infant length and head circumference with uncertain effects on intelligence in early life.[27,28] Malnutrition, however,

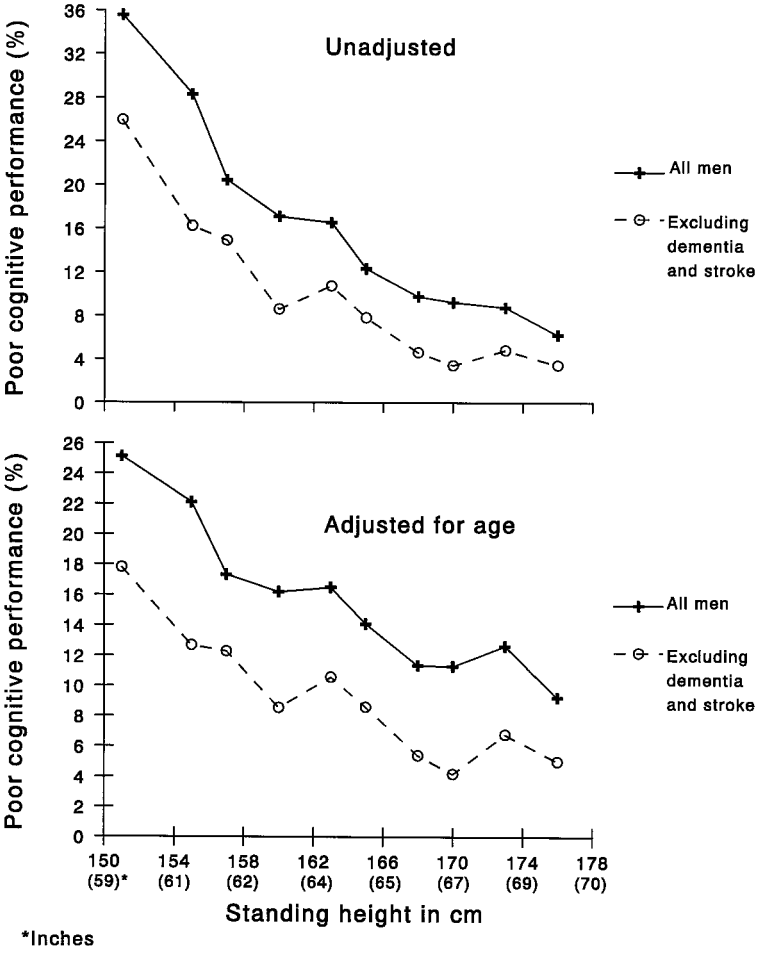


**Fig 1.** Unadjusted and age-adjusted rates of poor cognitive performance by height for all men and for men without dementia or stroke. In all instances, height was inversely associated with poor cognitive performance ($P < .001$).

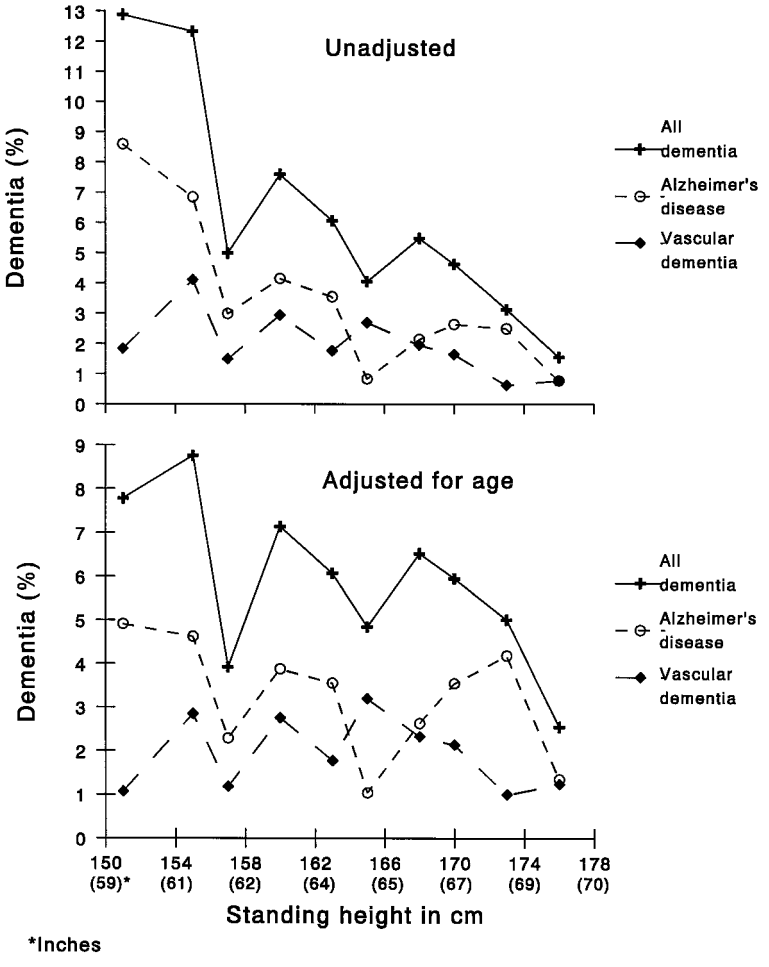


**Fig 2.** Unadjusted and age-adjusted rates of dementing illnesses by height. Without adjustment, height was inversely related to total dementia and Alzheimer's disease ($P < .001$). After age adjustment, the relation between height and total dementia and Alzheimer's disease is not statistically significant. Alzheimer's disease was significantly more common in men 154 cm (61 in) or shorter as compared with those who were taller ($P = .018$).

may be the most important modifiable determinant of stunting and early cognitive development.[26,29,30] Animal studies have also suggested that malnutrition has an adverse effect on specific regions of the brain, including the hippocampus,[31] an important structure for memory formation, and in humans, a region of the brain that is effected by early Alzheimer's disease. Low birth weight has further been related to deficits in cognition,[24] and small head size and short stature have been related to poor intelligence testing in schoolchildren.[29,30] In children without neurologic abnormalities, small head size at 8 months was associated with poor cognitive performance at 8 years of age.[29] It has also been noted that the impact of these deficits may be greatest in disadvantaged urban communities where rates of low birth weight infants are especially high.[24] Although some effects are thought to be reversible, even subtle failures in growth potential may have unknown long-term consequences on cognitive function later in life.

Although assessment of specific public health intervention programs to improve childhood and adolescent development needs encouragement, long-term effects on late-life cognition in areas in which childhood stress and deprivation are highest may be impossible to evaluate. Nevertheless, arguments have been made that prenatal and early life exposures could partially influence the clinical expression of Alzheimer's disease, particularly in those who are most vulnerable.[1,3,4] Others have argued that attention to nutrition and factors that promote growth should be extended to the adolescent as well.[32] Although attention to the progression of patterns of growth that include weight, height, and head circumference is important, it needs to be routinely accompanied by the often difficult task of identifying and altering nutrition and environmental factors that might deter growth potential within the family or community.[26,33]

Such attention could have real benefits if improvements can be made in the health and environment of high-risk children. Indeed, increases in food production and its distribution, along with improvements in medical care and quality of life, may be partly responsible for increases in human brain weight and head circumference that have been observed to occur during the century.[4,21,22] Data from Hawaii indicate that height is also increasing in younger birth cohorts. In addition, younger men in Hawaii had greater body mass index than those who were older, suggesting that increases in height throughout time are related to improved nutrition. Because poor nutrition and stress in childhood can influence height, brain growth, and cognitive capacities, one might suspect that these same childhood exposures, by virtue of their effect on attained brain reserves, could

also influence the expression of brain aging and dementing diseases in late life.[1–3,24–26,29,30]

Support for this latter possibility is based on the observation that education and occupation tend to have an association with the development of brain reserves and clinically apparent Alzheimer's disease.[1,2] Investigators from Seattle and New York observed that small head size was a determinant of the severity of Alzheimer's disease.[4,5] In Seattle, small head circumference was more common in poorly educated persons who lived their early lives in Japan and who were presumed to have migrated to the United States in an effort to improve their socioeconomic status. Similar phenomena could have occurred in the Honolulu cohort where a high proportion of subjects may have achieved a limited adult body and head size that cannot be entirely attributed to genetic predisposition. In a third report linking childhood intellectual development to late-life brain degeneration, investigators described an association between the density of neocortical neurofibrillary tangles at death and high verbal idea density in essays written on enrollment into a Catholic order more than a half century earlier.[3]

It is not clear how these factors relate to the declines in brain weight that progressively occur after 50 years of age.[22,23] Perhaps the effects of this decline on cognitive function are too subtle or harder to detect in individuals who have developed maximum brain reserves. Case detection in those with high levels of education or in those in highly skilled professions may also be more difficult because of delays in subtle pathologic changes that precede neurofibrillary tangles and neuritic plaques in Alzheimer's disease.

Of course, a definitive examination of the direct consequence of prenatal and early childhood development on late-life cognitive impairment is constrained by the inherent limits of any long-term epidemiologic study and by the lack of precision in identifying early childhood exposures in adult samples. More careful assessment would involve the follow-up of large cohorts of individuals over successive generations that bridge the gap between studies of cognition in childhood and adolescence with those that occur in the elderly. Conclusions from this report are only hypothetical and inferred from observations made in the Honolulu–Asia Aging Study in combination with data that link early nutrition and deprivation to childhood development[24–26,29,30] and with reports that link education and cognition in young adulthood to late-life memory and thinking impairment.[1–3]

Even within studies, however, conclusions are often subjected to flaws because of bias, self-selection, and imprecision. In the Honolulu–Asia Aging Study, short height in middle life may not be the best marker of brain size. Although height reflects genetic constitution and growth throughout the first 2 decades of life, neural capacity is thought to be almost entirely determined by the end of the first decade. Height, however, is still known to be correlated with brain weight.[22,34]

Education, father's occupation, and childhood residence also suffer from deficiencies as surrogates of childhood exposures that might influence growth and development. These factors were selected for analysis in this report because they seemed to be the most logical markers of childhood exposures that were available. Of course, it would have been more interesting to have interviewed the parents of the cohort members with regard to nutrition and childhood conditions. Although this might have been feasible when the Honolulu Heart Program began in 1965, it might have been possible in only a select group of surviving or available parents whose recall of conditions that may have existed at the beginning of the century would be uncertain.

Other than age and education, late-life cognitive function seems to have a stronger relationship with height than with the other exposure factors considered in this report. After adjustment for age, body mass index, years of childhood lived in Japan, level of education, and father's occupation, an association between height and cognitive performance remained statistically significant ($P = .001$). Here, the adjusted prevalence of poor cognitive performance in the shortest men was 20% vs 12% in the tallest men. Associations continued to persist after excluding men with dementia and stroke ($P = .002$). Among the other factors, only years lived in Japan was positively and independently related to poor cognitive function ($P = .004$). Although the relationship seemed weaker as compared to height, it suggests that there may have been other environmental conditions associated with living in Japan during childhood in the early part of the century that could have resulted in suboptimal growth and late-life cognitive impairment.

Although height seems to be related to cognitive performance in the elderly in the Honolulu–Asia Aging Study, its relationship with specific dementing illnesses is less clear, possibly because of small numbers. Height has a stronger relationship with Alzheimer's disease than with vascular dementia. Vascular dementia, however, most often involves a series of discreet and abrupt drops in cognitive function thought to be related to brain infarction. In contrast, Alzheimer's disease tends to progress slowly with beginnings in the entorhinal cortex and proceeding to the hippocampus and other regions of the brain, particularly the cerebral cortex. Although both forms of dementia may become clinically apparent only after functional or integrative thresholds are reached, the influence of preexisting brain reserves, as influenced by childhood exposures that affect growth, might have a greater role in the pathogenic process involving Alzheimer's disease.

An important factor to note in the Honolulu–Asia Aging Study is that participants are quite short. Average height in the Hawaii cohort is 8 to 10 cm (3–4 in) less than the average height of men of similar age in the general United States population, and it is not uncommon for a Japanese man in this cohort to be under 152 cm (60 in) tall. Although cognitive func-

tion seemed to improve across the full range of increasing heights, differences in the percentages of men with poor cognitive performance between adjacent heights seemed to be greater in the shorter height ranges than in the taller height ranges.

Whether our results apply to other ethnic groups or generations is unknown. The association between height and late-life cognitive performance could be harder to detect in samples comprised of taller individuals. Replication of findings from Hawaii in other population-based samples may require the study of persons of small stature or those who experienced deprivation during childhood. Observed differences across populations in the age-specific prevalence of cognitive impairment and Alzheimer's disease, such as the apparent differences between findings from Honolulu,[13] Framingham,[35] and East Boston,[36] might also be partially a result of differences in childhood experiences and nutrition.

Data from Hawaii are consistent with the possibility that generations which preceded the Honolulu cohort could have been more vulnerable to greater rates of late-life cognitive impairment because of their being shorter than current generations or because of exposure to environmental factors while living in Japan. Short life expectancy among earlier generations, however, might have prevented high rates of cognitive impairment from occurring. Data further suggest that as height increases and childhood development improves in younger birth cohorts, the prevalence of cognitive impairment and dementia in late life could decline in future years. Improvements in education might also contribute to this decline.[1] Increases in life expectancy, however, and survival of low birth weight infants without attention to neonatal growth or to factors that promote childhood and adolescent development could reverse this trend.[29] Our observations support the hypothesis that suboptimal nutrition and growth during childhood contribute to the development of cognitive impairment and dementing disease in late life and that dementia and cognitive impairment in the elderly may be partially preventable diseases.

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## WHY HMOs NOW LOVE REGULATION

Twenty-five (25) of America's largest health maintenance organizations (HMOs) endorsed legislation that would subject their industries to more government regulation. A response to political pressure? Not likely. Instead, it is an example of how large corporations use government to protect what they have rather than risk it in a truly competitive market.

Managed care is dying. HMOs such as Aetna, Kaiser, and Oxford are losing money because they are no longer able to dictate to consumers which health care services they can have. Faced with fed-up consumers, HMOs will, like the Big Three auto makers did in the 1980s, reluctantly restructure—becoming more responsive than before, but with a significantly smaller market share. Nothing can prevent this consumer-driven shakeup in health care.

Nothing, that is, except the HMO regulations Congress claims will protect consumers. The proposed regulations—mandating easier access to emergency care, drugs, specialists, and physicians not in HMO networks—will raise the cost of running a health plan. The likely result: more mergers, bigger HMOs, and more market concentration. More important, higher costs will crush HMOs' competition—entrepreneurial ventures that are responding to consumer demand for quality care.

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Noted by JFL, MD